IJSPT

ORIGINAL RESEARCH

THE CONSTRAINING EFFECT OF THE LATERAL FEMORAL INTERMUSCULAR SEPTUM ON PASSIVE HIP ADDUCTION IN UN-EMBALMED CADAVERS

Gesine H. Seeber, PT, MS[1,2]
Micah Lierly, PT, DPT[1]
Cameron Bassett, PT, DPT[1]
Christian Douthit, MD[1]
Mark Wilhelm, PT, DPT[3]
Anja Matthijs, PT, MS[1,4]
Omer Matthijs, PT, ScD[1,4]
Djordje Lazovic, MD[2]
Jean-Michel Brismée, PT, ScD[1]
Kerry Gilbert, PT, ScD[1]
Phil Sizer Jr., PT, PhD[1]

ABSTRACT

Background: Due to the lack of verifiable iliotibial band elongation in response to stretching, the anatomical, biomechanical, and physiological responses to treatment of iliotibial band syndrome remain unclear. The lateral intermuscular septum, consisting of multiple myofibroblasts, firmly anchors the iliotibial band to the femur.

Purpose and Hypothesis: The purpose of this in-situ study was to examine the constraining effect of the lateral intermuscular septum on available passive hip adduction range of motion in un-embalmed cadavers. It was hypothesized that an iliotibial band-septum-complex release would significantly increase passive hip adduction

Design: Within-specimen repeated measures in-situ design.

Setting: Anatomy laboratory.

Methods: Metal markers were inserted into selected anatomical landmarks in eleven (11) un-embalmed human cadavers. With the specimen supine, the test-side lower limb was passively adducted until maximum passive hip adduction was reached. This movement was repeated three times each within two conditions: (1) band-septum-complex intact and (2) band-septum-complex dissected. Digital video of marker displacement was captured throughout each trial. Still images from a start and an end position were extracted from each video sequence. A custom Matlab program was used to calculate frontal plane hip adduction angle changes from obtained images.

Results: Mean change in passive hip adduction after band-septum-complex release was -0.3° (SD 1.6°;95% CI: -1.33,0.76). A paired samples t-test revealed a non-significant difference ($t = -.611$; $p = .555$) in passive hip adduction for the band-septum-dissected condition (18.8 $\pm$ 3.9°) versus the band-septum-intact condition (18.5° $\pm$ 4.7°).

Conclusion: The lateral intermuscular septum does not appear to have a constraining effect on passive hip adduction in un-embalmed cadavers. Future research should evaluate the constraining effect of other selected tissues and conditions on hip adduction. Furthermore, inflammatory, metabolic, viscoelastic, and sensorimotor control properties within the iliotibial band in response to stretching should be investigated.

Level of Evidence: 3

Key words: Hip adduction, iliotibial band, lateral intermuscular septum, selected cutting.

[1] Center for Rehabilitation Research, Texas Tech University Health Sciences Center, Lubbock, Texas, USA.
[2] University Hospital of Orthopaedics and Trauma Surgery Pius-Hospital, Medical Campus University of Oldenburg, Oldenburg, Germany.
[3] Tufts University School of Medicine, Medford, MA, USA.
[4] Boma, Physical Therapy Outpatient Clinic, Mariazeller Straße 30, 8605 Kapfenberg, Styria, Austria.

Conflict of Interest: None to declare.
Funding: There was no funding received for this study.
Medical devices: No medical devices have been used in this study.

CORRESPONDING AUTHOR

Phillip S. Sizer Jr. PT, PhD, OCS, FAAOMPT
Professor & Program Director, ScD Program in PT
Director, Clinical Musculoskeletal Research Lab,
Center for Rehabilitation Research
Faculty, PhD Program in Rehabilitation Sciences, SOAHS
Faculty, Medical Pain Fellowship Program, SOM
Texas Tech University Health Sciences Center
3601 4th Street, Room 2B 138 - Mail Stop 6226
Lubbock, Texas 79430
Tel: 806.743.3902, Fax: 806.743.2189
E-mail: phil.sizer@ttuhsc.edu

DOI: 10.26603/ijspt20200042

INTRODUCTION

Iliotibial band syndrome (ITBS) is one of the most common causes of lateral knee pain in physically active individuals including runners, cyclists, tennis and football players or dancers.[1-4] The reported incidence is between 5% - 14% in runners[5] and 6% - 22% in army recruits.[1,2] Lavine et al[3] report that ITBS counts for 22% of all lower extremity overuse injuries. Iliotibial band syndrome frequently involves tissue inflammation and subsequent pain-induced functional limitations at the knee during sports and everyday activities. [5,6] However, etiology and treatment related to ITBS are currently being debated.

The iliotibial band (ITB) is a robust tissue,[7,8] representing the laterally thickened distal continuation of the tensor fascia lata. The longitudinal oriented ITB fibers are continuous with the fascia lata of the thigh, completely enclosing the lateral thigh. The ITB adheres firmly to the femoral linea aspera via the entire length of the lateral intermuscular septum (IMS), which divides the anterior and posterior thigh compartments, and extends distally between the vastus lateralis and biceps femoris muscles.[7,9-11] The IMS is anteriorly attached to the vastus lateralis. Posteriorly, the proximal quarter connects to the gluteus maximus and its distal three quarters to the short head of the biceps femoris.[7] Some of the distal ITB fibers contribute to the iliopatellar ligament, while the remaining fibers attach to the anterior-lateral tibial tubercle, also known as Gerdy´s tubercle.[9] Histologically, the ITB appears to be a tendon-like structure[12] with only a sparse number of elastic fibers.[10]

Iliotibial band syndrome is commonly thought to be caused by repetitive anterior-posterior motion of the deep ITB tissue over the lateral femoral epicondyle. This most frequently occurs at approximately 30° of knee flexion during cyclical flexion-extension movement, as found in walking and running activities.[13-18] However, other authors contradict this friction model and instead propose that medial-lateral movement of the ITB at approximately 30° of knee flexion results in compressive forces between a highly innervated fat pad deep to the ITB and the lateral femoral epicondyle.[10] Despite discrepancies, both suggested mechanisms lead to subsequent inflammatory processes of local tissues.[10,12,19]

Various non-surgical management strategies have been proposed for ITBS treatment,[13,18] including stretching, foam rolling, neuromuscular training, and manipulative treatment that include a strain-counter-strain technique and manual myofascial release techniques.[3,4,17,18,20-23] Stretching appears to be the most frequently suggested management strategy for reducing ITB dysfunction and symptoms.[4,17,20,21] It should be noted that treatment efficacy and tissue response to stretching has limited evidence supporting its value.[15]

Investigators have reported symptom improvement and increased hip adduction range of motion (as per the findings of the Ober-test) in response to clinical stretching exercises.[4,18,21] Yet, the anatomical, biomechanical, and physiological explanations for treatment response remain under debate.[2] Utilizing strain gauges in un-embalmed cadavers, Falvey et al[2] demonstrated that the ITB does not significantly elongate in response to a stretching maneuver, due to its firm longitudinal attachment to the femur. More recently, Wilhelm et al[8] demonstrated in fresh cadaveric ITB tissue in-vitro that the junction between tensor fascia lata and the ITB is the only ITB region that may exhibit significant deformation in response to a clinically relevant stretching load. Moreover, the authors were able to demonstrate that their clinical ITB stretching strategy did not result in appreciable mid-substance ITB tissue deformation.[8]

Consequently, symptom improvements in response to non-surgical treatment of ITBS must be related to another, yet unknown factor. Such findings could possibly be explained through deformation of the IMS versus the ITB, which has not been examined to date. The IMS' thin layer of dense irregular connective tissue originates from the deep lower limb fascia, coursing dorsal-medially and terminating on the linea aspera.[2,23] Selected authors suggest that a considerable share of myofibroblasts can be found in the deep lower limb fascia, potentially renduring this tissue responsive to stretching.[24,25] Moreover, Langevin et al[26] reported the mechano-transduction of fascial tissue, suggesting connective tissue's capacity to adapt to a tension force. Furthermore, van der Wal[27] concluded that a stretching regime designed to target a specific tissue in isolation could transmit to other connected, surrounding tissues. Hence, an ITB clinical stretch could influence IMS fibers, allowing those fibers to change the impact that stretching could subsequently have on ITB dynamics.

Cadaveric selective cutting has been previously used to examine the constraining effects of different tissues on selected mechanical properties or movements.[28-34] The selective cutting model in the present study was crafted in response to previous observations when piloting on cadaveric specimens for a different study in the thigh region. It was then that the investigators qualitatively observed increased hip adduction in response to an ITB-IMS complex release. This observation was however not quantified and had not been previously reported.

Therefore, the purpose of this in-situ study was to examine the constraining effect of the lateral IMS on available passive hip adduction range of motion in un-embalmed cadavers. Examining this effect will help determine the role of the IMS in ITBS and serves as a basis for further investigation into IMS mechanical response to treatment. The authors hypothesized that surgical release of the ITB conjunction with the lateral IMS would significantly increase available passive hip adduction in un-embalmed cadavers, testing the lateral IMS' role in constraining hip adduction and offer a testable mechanism possibly responsible for changed hip adduction after stretching.

METHODS

Research Design and Variables

The investigators implemented a prospective study using a within-specimen repeated measures design. The dependent variable was the available passive range of motion hip adduction. The independent variable used in this study was the ITB-IMS complex condition, which had the following two levels:

1. ITB-IMS complex intact

2. ITB-IMS complex separated

Subjects

The specimen sample comprised 11 fresh un-embalmed human cadavers (7 male and 4 female). Mean age at time of death was 70.5 (SD $\pm$ 12.8) years, ranging from 48 to 94 years. All cadaveric specimens used for this study were from the Texas Tech University Health Sciences Center gross anatomy laboratory. Cadaveric specimens were handled according to university policy and the State of Texas regulations defined by the Texas Tech University Health Sciences Center Anatomical Board. Specimen characteristics including comorbidities and individual cause of death are presented in Table 1. Data collection was completed using six right and five left lower limbs.

Table 1. *Study specimen characteristics.*

Subject	Sex	Age (years)	Comorbidities and Cause of Death
1	M	75	Congestive Heart Failure; Coronary Atherosclerosis
2	F	75	Non-Small Cell Lung Carcinoma; COPD
3	F	94	Acute Stroke; Peripheral Vascular Disease Atherosclerotic Heart Disease
4	M	76	Stage IV Melanoma with Brain Metastases
5	M	76	Embolic Infarcts of both Cerebral Hemispheres; Hypertension
6	M	70	Dementia; COPD; Diabetes Mellitus; Hypertension
7	F	48	Metastatic Pancreatic Cancer
8	M	69	COPD
9	M	72	Hemorrhagic Shock; Multi Organ Failure; Acute Respiratory Failure; CVA
10	F	72	Septic Shock; Urinary Tract Infection; Encephalopathy; Acute Renal Failure
11	M	49	Multi Organ Failure; Malignant Colon Tumor; Hypoglycemia

M = Male, F = Female, COPD = Chronic Obstructive Pulmonary Disease, CVA = Cerebrovascular Accident

All specimens fulfilled the following criteria:

(1) Bilateral intact full-length lower limbs

(2) No detectable abnormalities or damages to the pelvis, thigh, ITB, or IMS

(3) No known previous hip surgery

(4) No known current hip fractures

(5) No severe lower quarter tissue contractures.

Pre-Measurement Preparation

After placing the specimen supine on the examination table, the lower limb that was found most neutrally aligned during gross inspection was identified as the test-side lower limb. The rationale behind that was to visually identify obvious lower limb malalignment that may possibly have been caused from capsular or other soft tissue restrictions around the hip, and thus could have had a limiting impact on passive available hip adduction range of motion. Subsequently, threaded markers were inserted into each anterior superior iliac spine (ASIS) bilaterally and the test-side femur in order to compare the extent of passive available hip adduction with the ITB-IMS complex intact versus the ITB-IMS complex separated. Markers were placed in a standardized manner in the supine lying specimen. After the respective bony landmark was located through manual palpation, a countersink was used to create a starter hole for drill bit guidance. After pre-drilling, the commercially available drill bit (Black & Decker™, New BritIain, CT, USA) was removed, and a 3.5-inch Philips-head screw was implanted into the pre-drilled hole. Following the insertion of one marker into each ASIS, the first of two femoral Phillips-type screw markers was placed along the anterior midline 5 cm proximal from the patellar basis. In order to create a representative shank, a second femoral marker was inserted at the midpoint between the ipsilateral ASIS marker and the previously placed femoral marker. To maintain neutral rotation of the test extremity throughout marker placement, the intercondylar line of the femur was held in parallel alignment to the table surface.

Instrumentation

To objectively record maximum available passive hip adduction range of motion during all test conditions, digital motion recordings of the screw markers were captured using a commercial-quality high-resolution video camera (CANON XF305 HD, Canon Inc, Tokio, Japan). The camera was connected to a 21.5" LED CCTV monitor (ToteVision, Seattle, WA, USA). The video camera was mounted on a boom above the test table and aligned perpendicular to the testing plane in 1m-distance to the specimen. This distance was chosen in order to avoid contortion artifacts and ensure measurement validity. Video was recorded in high-definition with 1920-1080 pixels resolution and a frame rate of 50 frames per second in natural room illumination. Prior to each recording series, the 18x-zoom, 4.1-73.8mm lens was zoomed all the way out prior to zero back in on the specimen. The camera was set in autofocus mode during data capture.

Pre-Preparatory Procedures

Prior to marker insertion, both lower limbs were repeatedly moved in (1) hip abduction/adduction, flexion/extension, and internal/external rotation, and (2) knee flexion/extension for five minutes to reduce tissue stiffness and minimize any remnant muscle rigor. Subsequently, the supine-lying specimen was placed appropriately on the testing table and the screw markers were inserted to the ASISs and anterior femur as previously described.

With the screw markers in place and final camera set-up complete, the test-side lower extremity was moved passively through the frontal plane range to ensure that all important items were sufficiently captured on the video as witnessed in real-time on the LED monitor.

Data Collection Procedures

Throughout testing, the specimen´s torso was stabilized by one investigator who stood cranial to the specimen to ensure its position consistency. The opposite lower limb was placed in slight hip abduction in order to be able to move the test side lower limb through the whole passive available hip adduction range of motion. Two lines were constructed using the previously mentioned markers on determined anatomical landmarks. The first line was created between the markers at the anterior superior iliac spines ("ASIS line"). The second line was created between the two femoral markers ("Thigh line). The test-side lower limb was moved to an investigator-selected hip abduction angle greater than 90-degrees between the ASIS line and the thigh line to initiate data capture. Subsequently, the test-side lower limb was passively adducted towards the opposite limb until maximum available passive hip adduction was reached, determined by end of available passive movement indicated by pelvic motion. While moving the lower limb, care was taken that the intercondylar line was aligned parallel to the table surface to maintain lower limb neutral rotation. During movement, the heel was always maintained in slight contact with the table surface to avoid hip flexion. (Figure 1) This procedure was repeated three times with the ITB-IMS complex intact. Following the third trial, the ITB-IMS conjunction was carefully separated while preserving ITB integrity. The skin was incised, starting 5 cm distally from the ipsilateral ASIS, and stopped just proximal to the distal femoral marker. The entire lateral IMS was separated from the ITB starting at the distal end of tensor fascia lata (TFL) muscle and extending distally to the level of the distal femoral marker. After complete ITB-IMS separation was confirmed visually and via palpation, the skin was sewed back together. Following ITB-IMS junction transection, three passive hip adduction range of motions trials were performed in the same fashion as previously described. Digital motion recording was captured throughout each trial.

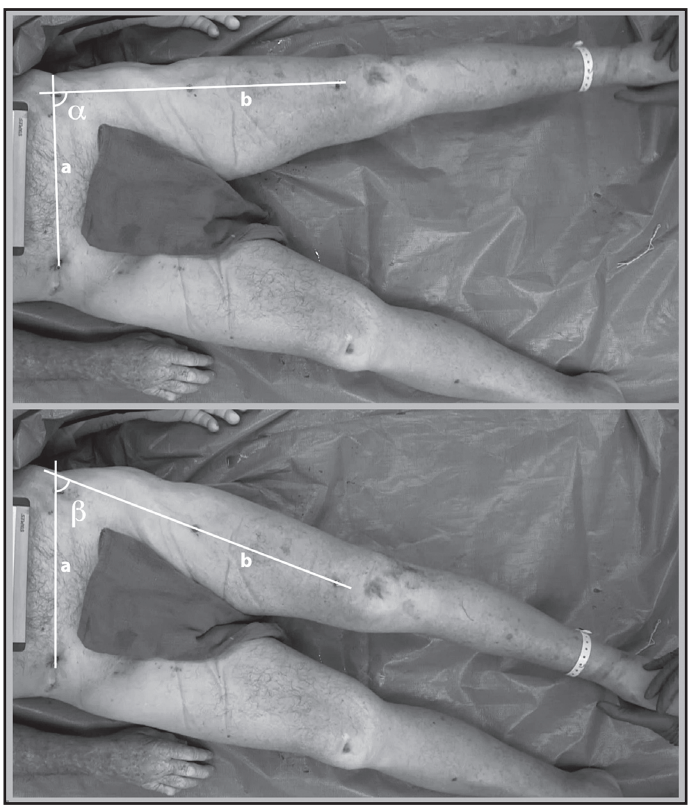

Figure 1. *Sample of passive available hip adduction measure with ITB-IMS-complex intact, a = ASIS line; b = thigh line; α = 90° angle between ASIS line and thigh line, starting position; β = angle between ASIS line and thigh line in maximum available passive hip adduction, end position.*

Image Digitization Process

Range of passively available hip adduction was defined by the change in the frontal plane angle created by the line between the ASIS line and the thigh line (Figure 1). To calculate this, still images of a hip adduction start and an end position were extracted from each captured video sequence. Predefined start position was a right (90 degrees) angle between the ASIS line and the thigh line. The end position was visually confirmed to be the maximum available recorded hip adduction position, where no further angular motion was detected on the video between the two lines with subsequent passive movement of the lower limb. Image digitization for data reduction purposes was conducted using a custom MATLAB program (Version R2016b; The Mathworks, Inc, Natwick, MA USA). This standardized reliable and valid uniplanar measurement procedure has been previously incorporated by several authors across different joint structures and tissues.[8,33,35-39] The MATLAB program prompted the user to select and import a baseline right angle image that represented the start position. In this image, four fixed points defined as the cross heads of the screw markers were chosen: starting with one point at the left ASIS followed by one point at the right ASIS, followed by one point on the proximal femur and one point on the distal femur. Each image was digitized three times in the exact same manner. The customized MATLAB program calculated the respective hip adduction angles for each digitization event, resulting in three values per image. A second image representing the end position of the same trial was subsequently selected and the procedure was precisely repeated and automatically transferred to an Excel spreadsheet. Hip adduction angle values of all three digitization events were used to calculate the mean hip adduction angle per image. The total change in hip adduction was calculated by subtracting the mean end position angle from the mean starting position angle and can be described with the following formula:

Passively available hip adduction =
mean start hip angle α– *mean end hip angle* β*

*see Figure 1 for depiction of angles

Data Analysis

Descriptive data analysis was calculated to summarize sample demographic characteristics. Moreover, values of central tendency (mean) and dispersion (standard deviation and 95% confidence intervals) for passive hip adduction range of motion for each condition (ITB-IMS separated versus ITB-IMS intact) were established. Data normality was defined as meeting at least two of the three following criteria: (1) Shapiro Wilk test p-value > 0.05, (2) skewness between -2 and +2, and (3) kurtosis between -2 and +2. A paired samples t-test was utilized to determine whether a difference in passive available hip adduction existed between the ITB-IMS-intact versus ITB-IMS-separated conditions. Significance was set at α = .05. All data analyses were performed using IBM SPSS Statistics (V23.0; IBM Corp. Armonk, NY, USA).

RESULTS

Specimen

A total of 11 cadavers were examined (Table 1). All provided cadavers fulfilled the inclusion criteria, so no specimen was excluded from the study.

Digital images

A total of 12 digital images per specimen were extracted from the digital recordings [3 trials x 2 ITB-IMS conditions (intact vs. separated) x 2 positions (start position vs. end position)]. All in all, 132 images were analyzed via MATLAB as described above.

Amount of passive available hip adduction

Passive available hip adduction in the ITB-IMS-intact condition was compared to the same movement in the ITB-IMS-separated condition. Mean change in passive available hip adduction range of motion after releasing the ITB conjunction with the lateral IMS was - 0.3° (SD 1.6°; 95% CI: -1.33, 0.76). Passive available hip adduction in the ITB-IMS-separated condition (18.8 ± 3.9°) was not significantly greater (t= -.611; p= .555) versus the ITB-IMS-intact condition (18.5° ± 4.7°; Figure 2).

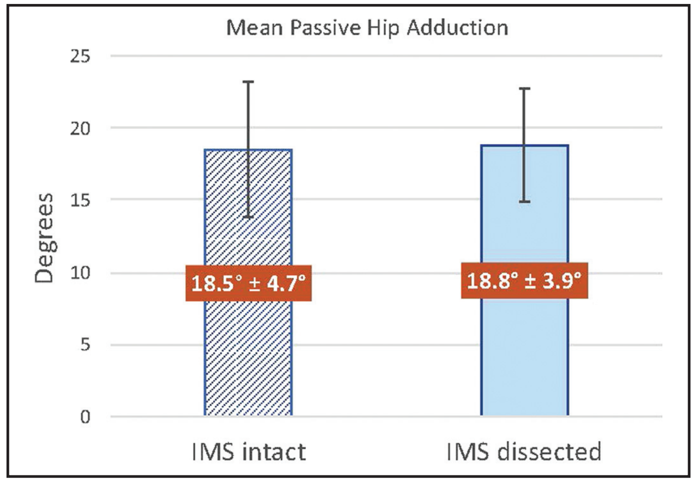

Figure 2. *Average passive hip adduction values (in degrees) with the lateral femoral intermuscular septum intact (IMS intact) versus dissected (IMS dissected).*

DISCUSSION

In the present study, separating the IMS from the ITB did not change the available range of passive hip adduction in un-embalmed cadavers. This was contrary to the investigator's experimental hypothesis, where an increase in hip adduction was expected. In ITBS patients, ITB flexibility often appears decreased, resulting in reduced hip adduction range of motion.[40,41] Increased ITB tightness adds to excess friction of the ITB over the lateral femoral epicondyle during cyclic knee motion in the sagittal plane, maintaining local tissue inflammation.[3,42] Applied therapeutic inputs through stretching exercises or foam roll use have been used for reducing ITB dysfunction and symptoms.[4,17,20,21] However, no plausible explanation has been offered as to why these interventions succeed.

Therapeutic interventions are reported to influence ITB flexibility as evidenced by Ober-test results.[41] Yet, limited evidence exists for the efficacy of any ITB stretching regimen.[15] A systematic review by Cheatham et al[43] suggest that self-myofascial release using a foam roll is beneficial for enhancing joint range of motion in various joints including the hip joint. Still, one must question the mechanical influence that such interventions have on ITB mechanical response. In a recent cadaveric study, Wilhelm et al[8] demonstrated that the primary ITB region that exhibits significant elongation in response to a clinically relevant stretching load was the proximal portion, which includes the TFL muscle.[8] Similarly, Falvey et al[2] explained the absence of elongation in response to a stretching maneuver due to the ITB´s firm longitudinal attachment to the femur via the IMS.

Even though the junction between the TFL and ITB is the only region of ITB complex that is appreciably extensible, patients report subjective symptom improvements, where increased hip adduction range of motion can be witnessed following clinical stretching exercises, foam-rolling or other (self-)myofascial release maneuvers to the entire complex.[4,18,21,43] Fredericson et al[20] considered that changes in different hip and thigh muscles (i.e. gluteal muscles, TFL, and vastus lateralis) might have added to increased hip adduction range of motion during stretching maneuvers meant to stretch the ITB versus actual changes in ITB length. However, they did not examine for other factors while keeping the hip movement in the frontal plane. In response, the current investigators examined the IMS' constraining effect on hip adduction mobility in the frontal plane.

As displayed in Figure 2 of the present study, the very small changes in mean hip adduction angle in response to IMS dissection were not statistically significant, which would most likely also not be clinically relevant at a difference of less than a degree. There are three possible explanations as to why an ITB-IMS conjunction release did not influence passive available hip adduction. First, the IMS attaches firmly to a large portion of the lateral femur.[7,9-11] While the IMS indirectly crosses the hip joint through the ITB attachment and then through the TFL, it does not directly cross the hip joint. Thus, it may not directly affect adduction range of motion. However, when the ITB-tensor fascia lata complex that has multiple tissue types (i.e. muscle and tendinous structures) is stretched, the force is likely dissipated through the pathway of least resistance.[8] In such a case, the TFL is likely not offering an appreciable amount of resistance, especially in un-embalmed cadavers with no muscle tone. So, when the hip was passively moved into adduction with the IMS intact, much of the lengthening likely occurred at the TFL level, concurring with previous findings.[8]

Secondly, authors have suggested that the ITB may not be the primary resistance to hip adduction.[2,44] These authors proposed that the superior portion of the hip capsule, as well as gluteus medius and minimus muscles, limit adduction range of motion to a greater extent than the ITB itself.[44] In a selected cutting study utilizing lightly embalmed cadavers with similar mobility to that seen in living persons, Willet and colleagues[44] witnessed results similar to the current study when cutting the ITB at midthigh level. Mean increase in hip adduction following ITB transection measured with a goniometer were 0.72° during the modified Ober test and 0.70° during the Ober test, respectively.[44] However, after transection of either the gluteus medius and minimus tendons or the hip joint capsule the authors found significant increase in hip adduction range of motion in both the modified Ober test and the Ober test.[44] Future research could examine the Willet et al effects further by selectively cutting the ITB first followed by the IMS. This could further test the influence of the IMS intended in the current study.

Third, it is unknown whether the cadaveric specimens utilized in the current study, as well as in the study of Willet et al[44] had been suffering from ITB tightness or pathology before death. This may have allowed other structures including the hip capsule to restrict adduction movement first, whereas in individuals with ITB pathology the ITB-IMS may have played a larger role in hip adduction restriction as suggested by other authors.[40,41]

Upon raw data examination, it was noted that some specimens lost a small amount of adduction movement once the IMS was dissected (Table 2). This phenomenon could have been the result of an ITB displacement in relation to the axis of rotation for hip adduction. Although, the values were all within a very small range of just one degree, thus having no effect on this study's overall outcome – that is that passive available hip adduction does not appreciably change in response to an ITB-IMS dissection in un-embalmed cadavers – these findings should be investigated further. Future research could examine the IMS´ constraining effect on sagittal plane ITB alignment and ITB length. Moreover, future studies could assess whether the IMS may have a constraining effect on hip adduction subject to different hip joint positions in the sagittal and transverse planes.

Table 2. *Values of each specimen's passive available hip adduction range of motion with the intermuscular septum dissected versus intact-condition.*

Specimen Number	Age	ITB-IMS intact	ITB-IMS cut	Intact-Cut difference
1	75	15	15.6	-0.6
2	75	20.5	19.8	0.7
3	94	23.8	24	-0.2
4	76	20.9	19.9	1
5	76	25.3	24.5	0.8
6	70	16.6	17.8	-1.2
7	48	9.9	14.1	-4.2
8	69	16.9	16.7	0.2
9	72	19.6	21.2	-1.6
10	72	22.3	21.3	1
11	49	13	12.2	0.8
Mean	70.5	18.5	18.8	-0.3

ITB-IMS intact = ITB conjunction with the lateral femoral intermuscular septum intact; ITB-IMS cut = ITB conjunction with the lateral femoral intermuscular septum dissected.

In the absence of any appreciable passive hip adduction changes in response to IMS dissection, one must further inquire into why clinically stretching an inextensible structure such as the ITB can effectively reduce ITBS symptoms and increase hip range of motion. Many different explanations are conceivable, however no sound evidence yet exists. First, one could suppose that stretching and foam-rolling do not have a mechanical influence on ITB fibers but rather influence the inflammatory state of the tissue. Acute inflammation is accompanied by an active resolution program, which starts within the first few hours after inflammation onset and involves the production of specialized pro-resolving mediators.[45,46] Researchers have suggested that stretching has a positive influence on the resolution of inflammatory processes within connective tissue.[47,48] Utilizing a carrageenan inflammation model in rats, Berrueta et al[47] observed that inflammatory processes decreased in vivo as well as ex vivo with both active and passive stretching compared to a no stretching condition. Besides a significantly decreased inflammatory thickness and cross-sectional area of the thoracolumbar fascia in their model, total and neutrophil cell counts within the inflammatory lesion were significantly smaller in stretched rats as compared to non-stretched rats. Moreover, the stretching induced increases in specialized pro-resolving mediators in vivo and ex vivo, suggesting a direct effect of stretching on the tissue.[47] This recently discovered interaction between musculoskeletal connective tissue and the immune system could potentially explain ITBS response to stretching and play a role in non-surgical ITBS treatment decisions.

The impact of stretching on ITB tissue metabolic changes may provide another plausible explanation for mechanical ITB interventions' effects.[49] Hotfield et al[49] examined changes on arterial blood flow and tissue perfusion after a series of self-myofascial release exercises using foam rolls. These authors demonstrated a hyper-perfusion in the ITB area, both immediately and 30 minutes after performing a foam rolling procedure. Blood circulation plays an essential role in tissue healing as it supports nutrients and oxygen supply.[50] Such a response could lend to tissue healing and pain reduction.

The third possible explanation for ITB stretching efficacy in ITBS patients centers on altered viscoelastic properties of the fascial tissue.[18,51] It is postulated that foam-rolling causes fascial tissue warming that results in increased pliability by transforming the tissue into a more fluid-like form and breaking up fibrous adhesions between the different fascial layers and thus restore soft-tissue extensibility.[51] Therefore, further study is merited regarding this concept.

A final possible explanation may center on fluid volume and pain response found in tissue underlying the ITB. It is possible that stretching and foam rolling could reduce vastus lateralis fluid volume, thus influencing the mechanical lever with respect to the hip axis of rotation. This may allow for a greater passive hip adduction before ITB resistance is met. Moreover, reducing fluid volume may influence pain by decreasing pressure associated with inflammation and mechanosensitivity. Thus, future investigations should further examine the influence of ITB stretching and foam-rolling on pro-inflammatory, metabolic, viscoelastic and fluid volume properties and responses to better explain treatment selection and response.

LIMITATIONS

One study limitation is found in its in-vitro design, where results from cadaveric investigations cannot be transferred to an in-vivo situation without critical considerations for missing information, such as muscle tone, contractile tension, and joint forces that are found in a living person. Moreover, in-vitro tissue properties may slightly differ from in-vivo tissue characteristics by virtue of their changes in mechanical properties. However, conducting this study would not be possible in-vivo due to its selective cutting design. Furthermore, the cadavers used for this investigation were un-embalmed in order to rule out major tissue alterations in response to embalming processes.

Furthermore, the study is limited by the age range of the specimens. Patients suffering from ITBS are usually from a younger population. In contrast, the cadavers used in this study were from an older population, making it challenging to transfer the results to younger individuals. However, younger cadaveric samples are difficult to obtain, hence investigators are often forced to use cadavers from older age groups.

Finally, the cadaveric specimens utilized in the current study were most likely not suffering from ITB tightness or pathology prior to death. This may have allowed other structures including the hip capsule to restrict adduction movement first, whereas in individuals with ITB pathology the ITB-IMS may have played a larger role in hip adduction restriction.

CONCLUSION

The results of the current study suggest that the lateral IMS does not have a constraining effect on passive hip adduction range of motion in un-embalmed cadavers. Future investigations should concentrate on evaluation of inflammatory, metabolic, viscoelastic, and fluid volume properties within the ITB and other selected tissues such as the vastus lateralis, gluteus medius and minimus musculotendinous tissue, and the hip joint capsule in response to an ITB stretching regime to better explain ITBS treatment selection and response.

REFERENCES

1. Sharma J, Greeves JP, Byers M, et al. Musculoskeletal injuries in British Army recruits: a prospective study of diagnosis-specific incidence and rehabilitation times. *BMC Musculoskelet Disord*. 2015;16(1):106.
2. Falvey EC, Clark RA, Franklyn-Miller A, et al. Iliotibial band syndrome: an examination of the evidence behind a number of treatment options. *Scand J Med Sci Sports*. 2010;20(4):580-587.
3. Lavine R. Iliotibial band friction syndrome. *Curr Rev Musculoskelet Med*. 2010;3(1-4):18-22.
4. Wang T-G, Jan M-H, Lin K-H, et al. Assessment of stretching of the iliotibial tract with Ober and modified Ober tests: an ultrasonographic study. *Arch Phys Med Rehabil*. 2006;87(10):1407-1411.
5. van der Worp MP, van der Horst N, de Wijer A, et al. Iliotibial band syndrome in runners: a systematic review. *Sports Med*. 2012;42(11):969-992.
6. Shamus J, Shamus E. The management of iliotibial band syndrome with a multifaceted approach: a double case report. *Int J Sports Phys Ther*. 2015;10(3):378-390.
7. Hayashi A, Maruyama Y. Lateral intermuscular septum of the thigh and short head of the biceps femoris muscle: an anatomic investigation with new clinical applications. *Plast Reconstr Surg*. 2001;108(6):1646-1654.
8. Wilhelm M, Matthijs O, Browne K, et al. Deformation response of the iliotibial band-tensor fascia lata complex to clinical-grade longitudinal tension loading in-vitro. *Int J Sports Phys Ther*. 2017;12(1):16-24.
9. Stecco A, Gilliar W, Hill R, et al. The anatomical and functional relation between gluteus maximus and fascia lata. *J Bodyw Mov Ther*. 2013;17(4):512-517.
10. Fairclough J, Hayashi K, Toumi H, et al. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. *J Anat*. 2006;208:309-316.
11. Kaplan EB. The iliotibial tract. Clinical and morphological significance. *J Bone Joint Surg Am*. 1958;40-A(4):817-832.
12. Baker RL, Souza RB, Fredericson M. Iliotibial band syndrome: soft tissue and biomechanical factors in evaluation and treatment. *Phys Med Rehabil*. 2011;3(6):550-561.
13. Beals C, Flanigan D. A review of treatments for iliotibial band syndrome in the athletic population. *J Sports Med*. 2013;2013(3):1-6.
14. Jelsing EJ, Finnoff JT, Cheville AL, et al. Sonographic evaluation of the iliotibial band at the

lateral femoral epicondyle: Does the iliotibial band move? *J Ultrasound Med*. 2013;32(7):1199-1206.

15. Ellis R, Hing W, Reid D. Iliotibial band friction syndrome-a systematic review. *Man Ther*. 2007;12(3):200-208.
16. Orchard JW, Fricker PA, Abud AT, et al. Biomechanics of iliotibial band friction syndrome in runners. *Am J Sports Med*. 1996;24(3):375-379.
17. Fredericson M, Wolf C. Iliotibial band syndrome in runners. *Sports Med*. 2005;35(5):451-459.
18. Strauss EJ, Kim S, Calcei JG, et al. Iliotibial band syndrome: evaluation and management. *J Am Acad Orthop Surg*. 2011;19:728-736.
19. Muhle C, Ahn JM, Yeh L, et al. Iliotibial band friction syndrome: MR imaging findings in 16 patients and MR arthrographic study of six cadaveric knees. *Radiology*. 1999;212:103-110.
20. Fredericson M, White JJ, MacMahon JM, et al. Quantitative analysis of the relative effectiveness of 3 iliotibial band stretches. *Arch Phys Med Rehabil*. 2002;83(5):589-592.
21. Fredericson M, Weir A. Practical management of iliotibial band friction syndrome in runners. *Clin J Sport Med*. 2006;16(261):261–168.
22. Noehren B, Davis I, Hamill J. ASB clinical biomechanics award winner 2006. Prospective study of the biomechanical factors associated with iliotibial band syndrome. *Clin Biomech*. 2007;22(9):951-956.
23. Pedowitz RN. Use of osteopathic manipulative treatment for iliotibial band friction syndrome. *J Am Osteopath Assoc*. 2005;105(12):563-567.
24. Schleip R, Kingler W, Lehman-Horn F. Fascia is able to contract in a smooth muscle-like manner and thereby influence musculoskeletal mechanics. In: Findley TW, Schleip R, eds. *Fascia Research. Basic Science and Complementary Health Care.* Munich; 2007.
25. Langevin HM, Huijing PA. Communicating about fascia: history, pitfalls, and recommendations. *Int J Ther Mass Bodywork*. 2009;2(4):3-8.
26. Langevin HM, Bouffard NA, Badger GJ, et al. Subcutaneous tissue fibroblast cytoskeletal remodeling induced by acupuncture: Evidence for a mechanotransduction-based mechanism. *J Cell Physiol.* 2006;207(3):767-774.
27. van der Wal J. The architecture of the connective tissue in the musculoskeletal system. In: Huijing PA, Hollander P, Findley TW, Schleip R, eds. *Fascia Research II. Basic Science and Implications for Conventional and Complementary Health Care.* Munich; 2009.
28. Smith MV, Costic RS, Allaire R, et al. A biomechanical analysis of the soft tissue and osseous constraints of the hip joint. *Knee Surg Sports Traumatol Arthrosc*. 2014;22(4):946-952.
29. Spratley EM, Wayne JS. Computational model of the human elbow and forearm: application to complex varus instability. *Ann Biomed Eng*. 2010;39(3): 1084-1091.
30. Park MC, Ahmad CS. Dynamic contributions of the flexor-pronator mass to elbow valgus stability. *J Bone Joint Surg*. 2004;86-A(10):2268-2274.
31. Ghosh KM, Hunt N, Blain A, et al. Isolated popliteus tendon injury does not lead to abnormal laxity in posterior-stabilised total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(6):1763-1769.
32. Dunning CE, Zarzour ZDS, Patterson SD, et al. Ligamentous stabilizers against posterolateral rotatory instability of the elbow. *J Bone Joint Surg*. 2001;83-A(12):1823-1828.
33. Seeber GH, Wilhelm MP, Windisch G,et al. Cadaveric evaluation of the lateral-anterior drawer test for examining posterior cruciate ligament integrity. *Int J Sports Phys Ther*. 2017;12(4):569-580.
34. Mitrogiannis L, Barbouti A, Kanavaros P, et al. Cadaveric-biomechanical study on medial retinaculum: Its stabilising role for the patella against lateral dislocation. *Folia Morphol (Warsz)*. 2018:1-6.
35. Gilbert KK, Brismee JM, Collins DL, et al. 2006 Young Investigator Award Winner: lumbosacral nerve root displacement and strain: part 1. A novel measurement technique during straight leg raise in unembalmed cadavers. *Spine*. 2007;32(14):1513-1520.
36. Gilbert KK, Brismee JM, Collins DL, et al. 2006 Young Investigator Award Winner: lumbosacral nerve root displacement and strain: part 2. A comparison of 2 straight leg raise conditions in unembalmed cadavers. *Spine*. 2007;32(14):1521-1525.
37. Lohman CM, Gilbert KK, Sobczak S, et al. 2015 Young Investigator Award Winner: Cervical Nerve Root Displacement and Strain During Upper Limb Neural Tension Testing: Part 1: A Minimally Invasive Assessment in Unembalmed Cadavers. *Spine*. 2015;40(11):793-800.
38. Lohman CM, Gilbert KK, Sobczak S, et al. 2015 Young Investigator Award Winner. Cervical Nerve Root Displacement and Strain During Upper Limb Neural Tension Testing. Part 2: Role of Foraminal Ligaments in the Cervical Spine. *Spine*. 2015;40(11):801-808.
39. Cobb SC, James CR, Hjertstedt M, et al. A digital photographic measurement method for quantifying foot posture: validity, reliability, and descriptive data. *J Athl Train*. 2011;46(1):20-30.

40. Grau S, Krauss I, Maiwald C, et al. Kinematic classification of iliotibial band syndrome in runners. *Scand J Med Sci Sports*. 2011;21(2):184-189.
41. Doucette SA, Goble M. The effect of exercise on patellar tracking in lateral patellar compression syndrome. *Am J Sports Med*. 1992;20(4):434-440.
42. Khaund R, Flynn SH. Iliotibial band syndrome: a common source of knee pain. *Am Fam Phys*. 2005;71(8):1545-1550.
43. Cheatham SW, Kolber MJ, Cain M, et al. The effects of self-myofascial release using a foam roll or roller massager on joint range of motion, muscle recovery, and performance: a systematic review. *Int J Sports Phys Ther*. 2015;10(6):827-838.
44. Willett GM, Keim SA, Shostrom VK, et al. An anatomic investigation of the Ober test. *Am J Sports Med*. 2016;44(3):696-701.
45. Serhan CN, Clish CB, BRannon J, et al. Novel functional sets of lipid-derived mediators with antiinflammatory actions generated from omega-3 fatty acids via cyclooxygenase 2–nonsteroidal antiinflammatory drugs and transcellular processing. *J Exp Med*. 2000;192(8):1197-1204.
46. Serhan CN. Pro-resolving lipid mediators are leads for resolution physiology. *Nature*. 2014;510(7503): 92-101.
47. Berrueta L, Muskaj I, Olenich S, et al. Stretching impacts inflammation resolution in connective tissue. *J Cell Physiol.* 2016;231(7):1621-1627.
48. Corey SM, Vizzard MA, Bouffard NA, et al. Stretching of the back improves gait, mechanical sensitivity and connective tissue inflammation in a rodent model. Baccei ML, ed. *PLOS Biology*. 2012;7(1):e29831-e29838.
49. Hotfield T, Swoboda B, Krinner S, et al. Acute effects of lateral thigh foam rolling exercises on arterial tissue perfusion determined by spectral Doppler and power Doppler ultrasound. *J Strength Cond*. 2017;31(4):893-900.
50. Lohman EB III, Bains GS, Lohman T, et al. A comparison of the effect of a variety of thermal and vibratory modalities on skin temperature and blood ow in healthy volunteers. *Med Sci Monit*. 2011;17(9):MT72-MT81.
51. MacDonald GZ, Penney MDH, Mullaley ME, et al. An acute bout of self-myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *J Strength Cond*. 2013;27(3):813-821.